Claims

1. A method for promoting axonal growth comprising treating a neuron with an NF-AT agonist.

2. The method of claim 1, wherein said NF-AT agonist interacts with calcineurin and increases the dephosphorylation of NF-AT.

3. The method of claim 1, wherein said NF-AT agonist binds NF-AT and increases its nuclear localization.

4. The method of claim 1, wherein the NF-AT agonist is calcineurin or an agent that upregulates the expression of calcineurin.

5. The method of claim 1, wherein the NF-AT agonist is an inhibitor of GSK-3.

6. The method of claim 5, wherein the inhibitor of GSK-3 is selected from the group consisting of an RNAi molecule, a ribozyme or a DNA enzyme that inhibits the expression of GSK3.

7. A method for promoting axonal growth comprising treating a neuron with a composition comprising an NF-AT agonist and another agent selected from the group consisting of: a neurotrophic factor, a neuropoietic factor, inosine, a fibroblast growth factor, an insulin-like growth factor, a platelet-derive growth factor, an anti-inflammatory, anti-NGF, anti-BDNF, anti-IGF-I, transforming growth factor-beta 1, other agents that increase production of inducible-nitric oxide synthase (i-NOS), an activator of macrophages, LPS, indomethacin, and a leukemia inhibitory factor (LIF).

8. A method for promoting axonal growth comprising administering an NF-AT agonist in a biodegradable nerve conduit.

9. A method to activate NF-AT dependent gene transcription comprising the use of a netrin or a neurotrophin.

10. A method to induce regeneration of neurons comprising treating said neurons with an NF-AT agonist.

11. The method of claim 10, wherein the NF-AT agonist enhances expression of NF-AT.

12. A pharmaceutical composition comprising an NFAT agonist and a pharmaceutically acceptable carrier.

13. A method of identifying a compound that is an NF-AT agonist and promotes axonal growth comprising:

(a) contacting the compound with a cell comprising NF-AT;

(b) determining the location of NF-AT within the cell in the presence and in the absence of the compound, wherein an increase of NF-AT in the nucleus indicates that the compound is an NF-AT agonist; and

(c) determining whether the compound promotes axonal growth.

14. A method of identifying a compound that is an NF-AT agonist and promotes axonal growth comprising:

(a) contacting a cell expressing NF-AT with a compound;

(b) determining the phosphorylation state of NF-AT in the presence and absence of the compound; wherein a decrease in the phosphorylation of NF-AT indicates that the compound is an NF-AT agonist; and

(c) determining whether the compound promotes axonal growth.

15. A method of identifying a compound that is an NF-AT agonist and promotes axonal growth comprising:

(a) contacting NF-AT with a phosphatase under conditions that allow the dephosphorylation of NF-AT in the presence and in the absence of a compound,

(b) determining the phosphorylation state of NF-AT, wherein an decrease in the phosphorylation indicates that the compound is an NF-AT agonist; and

(c) determining whether the compound promotes axonal growth.

16. The method of claim 15, wherein the phosphatase is calcineurin.

17. A method of identifying a compound that is an NF-AT agonist and promotes axonal growth comprising:

(a) contacting NF-AT with a kinase under conditions that allow the phosphorylation of NF-AT in the presence and in the absence of a compound,

(b) determining the phosphorylation state of NF-AT, wherein an decrease in the phosphorylation indicates that the compound is an NF-AT agonist; and

(c) determining whether the compound promotes axonal growth.

18. The method of claim 17 wherein the kinase is GSK-3.

19. A method of determining whether a compound is an NF-AT agonist comprising:

(a) transfecting a cell with an expression vector comprising a nucleic acid encoding a reporter gene operatively linked to an NF-AT dependent transcriptional regulatory sequence;

(b) incubating the cell in the presence and absence of a compound;

(c) measuring the expression of the reporter gene; wherein an increase in the expression of the reporter gene indicates that the compound is an NF-AT agonist; and

(d) determining whether the compound promotes axonal growth.